SETAC PRESS

Environmental Toxicology and Chemistry, Vol. 18, No. 6, pp. 1323–1328, 1999

Printed in the USA
0730-7268/99 $9.00 + .00

AN INVESTIGATION OF THE APPLICATION OF THE CANADIAN WATER QUALITY GUIDELINES

SHERRY L. WALKER and FRANK A.P.C. GOBAS*
School of Resource and Environmental Management, Faculty of Applied Sciences, Simon Fraser University, Burnaby, British Columbia, V5A 1S6, Canada

(*Received* 19 *March* 1998; *Accepted* 11 *September* 1998)

Abstract—The Canadian water quality guidelines for the protection of aquatic life assume that (1) the external water concentration is an effective measure of the concentration at the active site in organisms, which is ultimately responsible for a toxic response, and that (2) the safety factor accounts for any differences between laboratory and field conditions as well as the extrapolation from the effect concentration to a long-term no-effect concentration. This study examines these assumptions and assesses potential errors that environmental managers can make when applying the guidelines. The methodology is based on assessing the probability that internal concentrations of several contaminants are greater than or less than the "safe" concentration, assumed by the guideline, when the water concentration is at the water quality guideline. Results derived from empirical observations and a food-web bioaccumulation model show that a high probability (62–100%) exists that safe internal concentrations are exceeded for polychlorinated biphenyls, 1,2,4,5-tetrachlorobenzene, and hexachlorobenzene in Lake Ontario when the aqueous concentrations are at the water quality guideline values. This is due to field bioaccumulation factors being greater than the bioaccumulation factors in laboratory toxicity tests used for the water quality guideline development. Factors contributing to the exceedence of safe internal concentrations at the water quality guideline values are identified. Recommendations for improvement of the water quality guideline process are provided.

Keywords—Bioaccumulation Water quality objectives Food-chain model

INTRODUCTION

The Canadian water quality guidelines (WQG) are toxic effects-based environmental concentrations in water that are recommended to protect various resource uses (i.e., aquatic life, wildlife, recreation, agriculture, and drinking water) of these media. One of the goals of the guidelines for the protection of aquatic life is the protection of all life stages of all species [1]. The methodology generally used to achieve this goal is to estimate a "safe" concentration in water from available standard laboratory toxicity experiments for individual substances with species such as *Daphnia magna,* rainbow trout, and fathead minnows. In these experiments, the organisms are typically exposed to aqueous solutions of various fixed concentrations for a defined period of time, after which a toxicological endpoint, such as mortality, growth rate, or reproductive ability, is determined. A WQG is then derived by applying a safety factor to the most sensitive effect level (either the lowest observable effect level [LOEL] or LC50) for the most sensitive species reported in the toxicological literature, assuming that, by protecting this species, other species will be protected as well. The safety factor is used to account for differences in sensitivity among species, laboratory versus field conditions, test endpoints, and convert the effect concentration to a long-term no-effect concentration [1].

Two main assumptions are inherent in the WQG protocol for aquatic life. First, it is assumed that the external water concentration is an effective measure of the concentration at the organism's active site, which is ultimately responsible for the toxic response. Second, it is assumed that the safety factor is large enough to account for any differences between laboratory and field conditions, the extrapolation being from the effect concentration to a long-term no-effect concentration as well as any differences in species' sensitivities to the chemical substance. If these assumptions hold, the water quality guideline protocol can be expected to be protective as intended, and internal concentrations at the active site in organisms from the field will not exceed the level that was presumed to be safe from the laboratory toxicity data. However, if these assumptions are incorrect, one of two errors might occur. Environmental managers might assume that the safe internal concentration is not exceeded at the water quality guideline in the field when in fact it is and incorrectly conclude that actions to control the substance are unnecessary. This is analogous to a Type II error. Conversely, environmental managers might conclude that the safe internal concentration is exceeded at water concentrations above the water quality guideline and that further action to manage the substance is necessary when in fact it is not. This is analogous to a Type I error.

It is recognized by the Canadian Council of Ministers of the Environment [2] that the use of WQG requires an understanding of the chemical, physical, and biological characteristics of the water body as well as the behavior of the substance once it is introduced into the aquatic environment. In other words, the guidelines must be adjusted when deriving site-specific objectives so that the exposure concentration for a given substance is still an accurate surrogate for the concentration at the target site(s) of action. Factors that can modify toxicity to aquatic organisms—including temperature, dissolved oxygen, ionization, particulate matter, exposure, kinetics, mechanisms of toxicity, bioaccumulation, biomagnification, and biotransformations—are acknowledged by the Canadian Council of Ministers of the Environment [2]. However,

* To whom correspondence may be addressed (gobas@sfu.ca).

Table 1. The lowest observable effect level (LOEL) (μg/L) used in the Canadian water-quality guideline (WQG) development; the WQG (μg/L); the safety factor used in the WQG development process; the bioaccumulation factor $BAF_{L,Lab}$ (L/kg lipid) in the toxicity experiment used to derive the WQG; the bioaccumulation factor $BAF_{L,OBS}$ (L/kg lipid) observed in lake trout from Lake Ontario; the bioaccumulation factor $BAF_{L,MOD}$ (L/kg lipid) in lake trout from Lake Ontario estimated by the food-web bioaccumulation model; the safe internal concentration $C_{L,SAFE}$ (μg/kg lipid); the critical internal concentration $C_{L,CRIT}$ (μg/kg lipid); and the internal tissue concentration $C_{L,WQG}$ (μg/kg lipid) expected at the WQG for lake trout[a]

	PCBs	TECB	HCB
LOEL	0.2 [6]	86 [7]	3.5 [10]
WQG	0.001 [2]	0.15 [2]	0.0065 [2]
Safety factor	0.005 [2]	0.0025 [2]	0.0015 [2]
$BAF_{L,LAB}$	1.85×10^6 ($\pm 0.47 \times 10^6$)	5.01×10^4 ($+1.29 \times 10^4$) (-1.03×10^4)	1.66×10^5 ($\pm 0.63 \times 10^5$)
$BAF_{L,OBS}$	5.2×10^7 ($\pm 1.9 \times 10^7$)	3.1×10^5 ($\pm 1.6 \times 10^5$)	3.4×10^6 ($\pm 1.6 \times 10^6$)
$BAF_{L,MOD}$	8.0×10^7 (-4.1×10^7, $+8.2 \times 10^7$)	8.1×10^4 (-1.2×10^4, $+1.4 \times 10^4$)	2.6×10^6 (-1.1×10^6, $+1.8 \times 10^6$)
$C_{L,CRIT}$	3.70×10^5 ($\pm 0.94 \times 10^5$)	4.31×10^6 (-0.88×10^6, $+1.1 \times 10^6$)	5.81×10^5 ($\pm 2.2 \times 10^5$)
$C_{L,SAFE}$	1.9×10^3 ($\pm 0.47 \times 10^3$)	1.1×10^4 (-0.22×10^4, $+0.28 \times 10^4$)	8.71×10^2 ($\pm 3.30 \times 10^2$)
$C_{L,WQG}$ (observed)	5.2×10^4 ($\pm 1.9 \times 10^4$)	4.7×10^4 ($\pm 2.3 \times 10^4$)	2.2×10^4 ($\pm 0.99 \times 10^4$)
$C_{L,WQG}$ (model)	8.0×10^4 (-4.1×10^4, $+8.2 \times 10^4$)	1.2×10^4 (-0.18×10^4, $+0.21 \times 10^4$)	1.7×10^4 (-0.72×10^4, $+1.2 \times 10^4$)

[a] References are given in square brackets; standard deviations are in parentheses. Lower and upper standard deviations that are unequal refer to data that are log normally distributed.

no specific quantitative guidance currently exists for the incorporation of these site-specific factors into the development of site-specific objectives for organic substances, and resource managers typically adopt these guidelines directly to assess and manage water quality.

In this study, we investigate the situation in which the Canadian water quality guidelines are applied directly. The purpose of this study is twofold. First, we explore the applicability of the assumptions inherent in the water quality guideline protocol and assess the magnitude of Type I and Type II errors if the current water quality guidelines for the protection of aquatic life are applied directly to assess and manage environmental quality on a site-specific basis. To investigate these errors, we examine the differences in the relationship between the water concentration and internal concentration under laboratory and field conditions. Second, we propose and illustrate simple methods that can be used to improve the use of water quality guidelines by incorporating site-specific factors and processes that affect the relationship between concentrations of chemicals in water and aquatic biota.

METHODOLOGY

Summary

Our methodology uses four steps. First, we determine the "critical" and the "safe" internal concentrations for selected substances from the laboratory toxicity test results that were used to derive the WQG. The critical concentration is the concentration of the substance (in units of grams chemical per kilogram [wet weight] body weight) that is present in the organism when the toxic effect is observed in the toxicity experiment. The safe concentration is the concentration of the chemical in the organism that can be viewed as the concentration that is considered to be acceptable according to the guideline. It is the critical concentration multiplied by the safety factor (SF). Second, we derive bioaccumulation factors (BAFs) of the same compounds at an actual field site using (1) reported, observed data for water and biota and (2) a food-web bioaccumulation model. Third, we apply a hypothetical scenario in which the water concentration at the field site is assumed to be at the recommended WQG. The concentrations in the organisms in the field are then determined by multiplying the water quality guideline and the BAF and compared against the safe and critical concentrations. Type I and Type II errors are then assessed. Values given in parentheses are standard deviations of the mean. Lower and upper standard deviations that are unequal refer to data that are lognormally distributed. Standard deviations were either obtained from reported values or derived through simple error analysis.

Selection of substances

Three substances were selected to examine the relationship between the water concentration and internal concentration in the laboratory and field: polychlorinated biphenyls (PCBs), 1,2,4,5-tetrachlorobenzene (TECB), and hexachlorobenzene (HCB). These chemicals were selected because of the availability of water quality guidelines, measured environmental concentration data, and well-established bioaccumulation properties. The respective recommended Canadian water quality guidelines and lowest observed effect levels (LOEL) from which the guidelines were derived are listed in Table 1.

Step 1: Derivation of the critical and safe internal concentrations

For each chemical substance, we determined a critical concentration in the organism at the LOEL during the laboratory experiments used to develop the WQG. This was done by (1) using measured internal concentrations associated with the observed toxic effects, (2) multiplying measured bioconcentration factors by the LOEL to predict the internal concentrations, or (3) applying bioconcentration models [3–5] to assess internal concentrations during the toxicity test from which the guideline was developed. The safe internal concentration was then derived by multiplying the critical concentration and the safety factor used in the WQG development process. This

assumes that the safety factor used to develop the WQG for exposure-based toxicity data is appropriate for determining safe internal concentrations for aquatic organisms.

Polychlorinated biphenyls. To predict the critical internal concentration of PCBs associated with the LOEL, we used the results from the multigeneration toxicity and bioaccumulation experiment [6] of Arochlor 1248 in fathead minnows that was used to derive the WQG. Because DeFoe et al. [6] provided a graph showing a regression line of concentrations of Arochlor 1248 in fish lipid at various exposure concentrations in water, we derived a linear regression equation (standard deviations are reported in parentheses), that is, $C_L = 1.85\ (\pm 0.47)\cdot 10^6 \cdot C_W$, for the concentration of Arochlor 1248 in the fish lipid (C_L, g/kg) for various exposure concentrations in water (C_W, g/L). This regression equation was then used to derive the concentrations of total PCBs in fish lipid (the critical concentration) corresponding to the LOEL of 0.2 μg/L. The safe internal concentration then followed by multiplying the critical concentration by the safety factor of 0.005.

1,2,4,5-Tetrachlorobenzene. To derive the critical internal concentration for TECB from the LOEL reported for 50-mg American flagfish (*Jordanella floridae*) [7], a lipid-based (BCF_L) bioconcentration factor for TECB in this fish species was estimated from the lipid–water partition coefficient K_L, that is, $10^{4.7(\pm 0.1)}$ L/kg lipid [8]. This method's assumption that steady state was achieved in the 28-d exposure period of the toxicity experiment is supported by results [9] illustrating that the time to reach 90% of steady state is 5.71 (±0.49) and 7.77 (±0.88) d for whole fish and fish lipid, respectively, for larger individuals of 0.5 to 4 g. The critical and safe internal concentrations were then derived as

$$C_{L,CRIT} = BCF_L \cdot LOEL \quad (1)$$

$$C_{L,SAFE} = BCF_L \cdot WQG = C_{L,CRIT} \cdot SF \quad (2)$$

Hexachlorobenzene. The internal HCB concentration in largemouth bass (*Micropterus salmoides*) at the LOEL of 3.5 μg/L [10] and the WQG of 0.0065 μg/L during a 10-d exposure period after which toxic effects were observed was estimated by deriving the non-steady-state bioconcentration factor (L/kg lipid) after 10 d, $BCF_{L,10}$, as

$$BCF_{L,10} = [k_1/(k_2 \cdot L)] \cdot [1 - \exp(-k_2 \cdot t)] \quad (3)$$

where the uptake rate constant k_1, that is, 1,996 (±738) L/kg fish/d, was derived through linear regression of observed concentration data [10] during a linear uptake; the elimination rate constant k_2, that is, 0.12 (±0.02)/d, was determined through linear regression of the ln(concentration in the fish) versus time data during the elimination experiment [10]; the lipid content L is 0.07 and t is 10 d. The critical internal concentrations were then derived as the product of $BCF_{L,10}$ and the LOEL, and the safe concentration followed as the product of the critical concentration and the safety factor (Table 1).

An overview of the safe and critical internal concentrations associated with the water quality guidelines are presented for all the test chemicals in Table 1.

Step 2: Derivation of bioaccumulation factors in the field

Actual bioaccumulation factors (Table 1) of PCB, TECB, and HCB in Canadian waters were taken from Oliver and Niimi [11,12], who reported empirical BAF_L for pontoporeia, oligochaetes, sculpin, alewife, smelt, and lake trout in Lake Ontario. In addition, a stochastic food-web bioaccumulation model [5] was used to predict the BAF_L from reported [11] water and sediment concentrations for Lake Ontario. The bioaccumulation model combines the toxicokinetics of chemical uptake, elimination and bioaccumulation in individual organisms, and the trophodynamics of contaminants in the food web. The model assumes that steady state is achieved between the organisms and the concentrations in the water and sediment. The model was tested for a large range of organic substances in Lake Ontario [5]. The advantage of the model is that it can assess the value of BAF_L at different sites and under various environmental conditions.

Step 3: Deriving internal concentrations in the field at the WQG

Assuming that the water concentration is at the level of the WQG, the internal concentration in the organism at the WQG ($C_{L,WQG}$) can be assessed on the basis of the empirically and/or model-derived BAF_L because

$$C_{L,WQG} = BAF_L \cdot WQG \quad (4)$$

Any uncertainty in the BAF_L is directly reflected in $C_{L,WQG}$, as the value for the WQG does not include uncertainty.

Step 4: Measurement of Type I and Type II errors

A Type I error is defined as a situation in which, under field conditions, the concentration in biota is less than the mean safe internal concentration in the lipid when the chemical concentration in the water is at the WQG. In this situation, an environmental manager might assume that aquatic biota are at risk and/or decide that further monitoring or controls are necessary when in fact risks to aquatic biota are negligible because the safe internal concentration is not exceeded. A Type II error is defined as a situation in which, under field conditions, the concentration in biota is greater than the mean safe internal concentration in the lipid while the chemical concentration in water is at the WQG. In this case, it might be assumed that the risk to biota is negligible and that remediation efforts are not warranted when in fact a substantial risk exists and remediation efforts are appropriate. The Type I and Type II errors are determined by comparing the distribution of $C_{L,WQG}$ to $C_{L,SAFE}$ (Fig. 1). A Type I error is characterized as the proportion of the distribution of $C_{L,WQG}$ less than $C_{L,SAFE}$. A Type II error is determined as the proportion of the distribution of $C_{L,WQG}$ exceeding $C_{L,SAFE}$. In addition, we determined the proportion of the $C_{L,WQG}$ distribution exceeding the critical concentration ($C_{L,CRIT}$) (Fig. 1).

RESULTS

A compilation of field- and laboratory-derived bioaccumulation factors for PCB, TECB, and HCB as well as safe and critical internal tissue concentrations and tissue concentrations at the WQG are presented in Table 1. Bioaccumulation factors derived for PCBs, TECB, and HCB in Lake Ontario using either measured concentration data or a food-web bioaccumulation model were considerably larger than those determined for the laboratory experiments used in the WQG development process. As a result, concentrations in Lake Ontario biota ($C_{L,WQG}$) can be expected to be greater than the safe internal concentration ($C_{L,SAFE}$) if the water concentration of the chemical substance is at the WQG (Fig. 2). Figure 2 and Table 2 illustrate that when considering uncertainty in the estimation of $C_{L,SAFE}$ and variability in estimation of $C_{L,WQG}$, the probability is high that the mean safe internal concentration

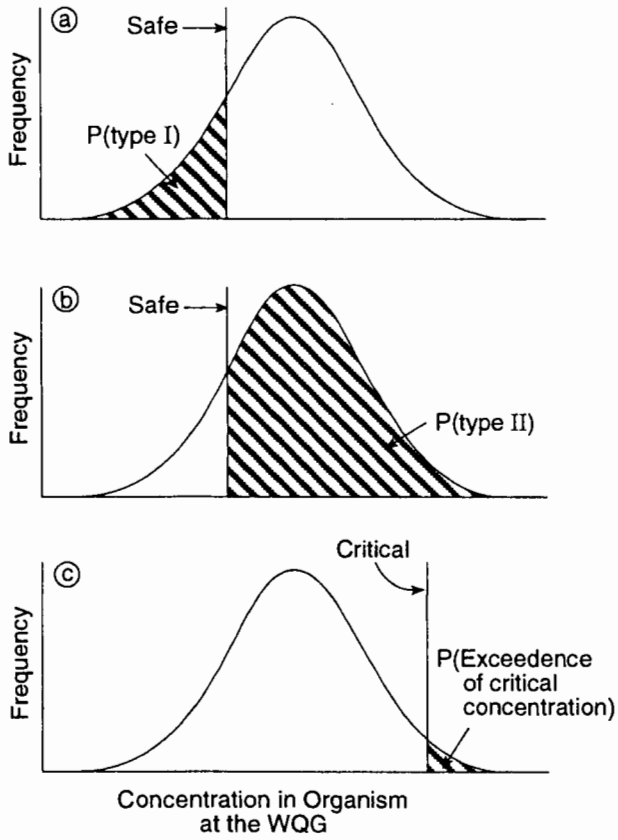

Fig. 1. Schematic diagram illustrating the comparison of internal tissue concentrations (g/kg lipid) at the water quality guideline to the internal concentration ($C_{L,SAFE}$) that is designated to be safe (a, b) and critical (c) in the water quality guideline development process.

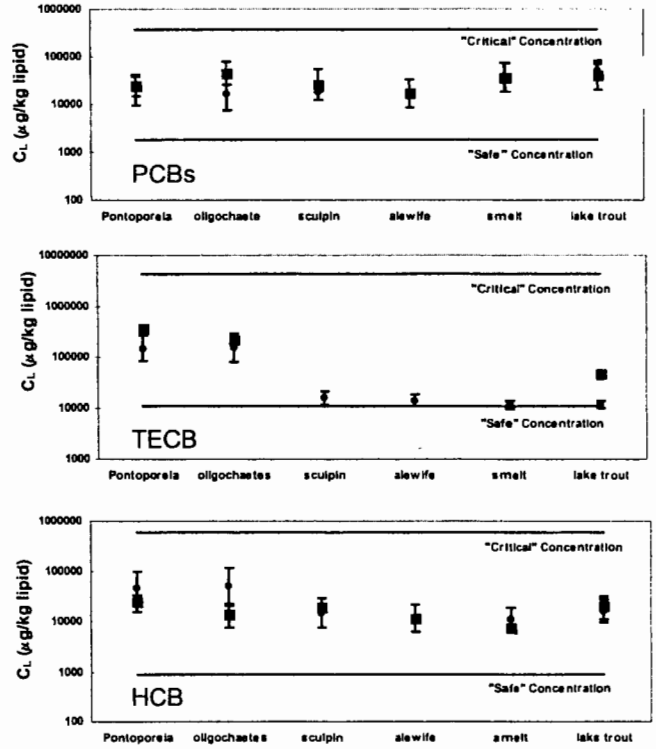

Fig. 2. Expected internal tissue concentrations (µg/kg lipid), derived from empirical data (●) and model calculations (■), of polychlorinated biphenyls (PCBs), 1,2,4,5-tetrachlorobenzene (TECB), and hexachlorobenzene (HCB) in several Lake Ontario organisms when the aqueous concentration is at the water quality guideline in relation to the internal concentration ($C_{L,SAFE}$) that is designated to be safe (lower solid line) and critical (higher solid line) in the water quality guideline development process.

is exceeded if the water concentration is at the WQG in the populations of all six species in Lake Ontario for all three chemical substances. The expected exceedence is the greatest for the very hydrophobic substances (PCBs and HCB). Table 2 illustrates that this corresponds to a high (62–100%) probability of committing a Type II error. The probabilities of not meeting the mean safe C_L (a Type I error) were relatively low (Table 2). The probabilities that the critical concentration ($C_{L,CRIT}$) is exceeded were low (0–6%).

Because the model-derived BAF_L data are in good agree-

Table 2. Probabilities (%) of committing Type I and Type II errors and the probability (%) of exceeding the critical internal concentration in various species of Lake Ontario for polychlorinated biphenyls (PCBs), 1,2,4,5-tetrachlorobenzene (TECB), and hexachlorobenzene (HCB) as derived from empirical data (observed) and model calculations (model)[a]

		PCBs		TECB		HCB	
		Observed	Model	Observed	Model	Observed	Model
Type II error ($C_{L,WQG} > C_{L,SAFE}$)	Pontoporeia	94	100	93	100	100	100
	Oligochaete	95	100	91	100	99	100
	Sculpin	100	100	NA	88	100	100
	Alewife	100	100	NA	85	100	100
	Smelt	100	100	NA	62	100	100
	Lake trout	99.8	100	96	73	95	100
Type I error ($C_{L,WOG} < C_{L,SAFE}$)	Pontoporeia	6	0	7	0	0.4	0
	Oligochaete	5	0	9	0	1.2	0
	Sculpin	0	0	NA	12	0	0
	Alewife	0	0	NA	15	0	0
	Smelt	0	0	NA	38	0	0
	Lake trout	0.2	0	5	27	5	0
Exceeding critical concentration ($C_{L,WQG} > C_{L,CRIT}$)	Pontoporeia	0	0	0	0	0	4
	Oligochaete	0	0	0	0	0	6
	Sculpin	0	0	NA	0	0	0
	Alewife	0	0	NA	0	0	0
	Smelt	0	0	NA	0	0	0
	Lake trout	0	0	0	0	0	0

[a] NA = Not available.

ment with those derived from empirical data in Canadian waters (Table 1), no substantial differences exist in the exceedence of $C_{L,WQG}$ over the $C_{L,SAFE}$ when using empirical data or model calculations. The probability for values of $C_{L,WQG}$ to be greater than $C_{L,SAFE}$ is slightly greater when assessed through the food-web model calculations than that derived using empirical data because the model predicts lognormally distributed concentrations, resulting in a larger percentage of higher concentrations in biota, whereas the measured concentrations were reported to be normally distributed.

DISCUSSION

The results illustrate that it is possible that aqueous chemical concentrations at the water quality guideline result in internal tissue concentrations in fish and benthic invertebrate species that are much greater than the concentrations anticipated in the guideline development process. This implies that the discrepancy between the water–internal organism concentration relationships (or BAF_L), due to toxicokinetic factors, accounts for a large part of the safety factors that are used in the derivation of the guidelines. For example, for PCB the safety factor that is applied in the WQG development process is 200 (or 1/0.005), and the $C_{L,WQG}$ in lake trout exceeds $C_{L,SAFE}$ by a factor of 27, leaving a factor of approx. 7 to account for toxicodynamic factors related to differences and variability in inter- and intraspecies sensitivity, the extrapolation being from the effect concentration to a long-term no-effect concentration and other sources of uncertainty, such as the occurrence of the chemicals in mixtures with other contaminants. The latter can have important management implications because it can be assumed that aquatic biota are adequately protected at the recommended water quality guideline and concluded that further management action is unnecessary when in fact the biota are at considerable risk. This is especially relevant in cases in which safety factors are small, as in the case of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin, in which the internal tissue concentration at the LOEL (or $C_{L,CRIT}$) of 0.182 μg/kg lipid [13] is very close (a factor of 3.6) to the proposed draft tissue residue guideline (or $C_{L,SAFE}$) of 0.05 μg TEQ/kg lipid [14] associated with the draft WQG of 0.06 pg TEQ/L [14]. In these cases, a small underestimation of the bioaccumulation factors can actually result in field situations in which the internal concentration at the WQG exceeds the internal tissue concentration at the LOEL.

Water–internal organism concentration relationships vary considerably between laboratory toxicity tests and the actual environment for several reasons. First, many hydrophobic organic chemicals are absorbed by aquatic organisms in the environment from water and food, whereas in the chronic toxicity tests, from which the guidelines were derived, the test species are often exposed only to waterborne chemicals. It has been shown that, for the test chemicals in this study, the diet can be the main source of chemical uptake for benthic organisms and fish in Lake Ontario, whereas chemical uptake from water is negligible [5]. Second, very hydrophobic organic chemicals, such as PCBs, HCB, and others (e.g., 2,3,7,8-tetrachlorodibenzo-*p*-dioxin), have the ability to biomagnify in food chains, resulting in concentrations in the organism of higher trophic levels that are much greater than those in the often smaller organisms used in toxicity experiments while exposed to the same concentration in the water. Third, the exposure duration in toxicity experiments are often much shorter than those in the actual environment. Therefore, it is possible that the internal tissue concentration of the organism in the toxicity test has not approached the steady-state levels to the same extent as that in organisms in the field that have been exposed for their entire lives. For example, for HCB steady state between internal and water concentrations for biota are approached in Lake Ontario, whereas steady state was not achieved in the 10-d laboratory toxicity test from which the guideline was derived. Fourth, sediment-water disequilibria control to a significant degree how much of the chemical is absorbed from the sediments via the benthic food chain in comparison to uptake from the water via the respiratory surface and/or the pelagic food chain. Sediment–water disequilibria can occur when chemical concentrations vary with time because of a number of factors, such as changes in chemical inputs and physical factors (e.g., changes in water flow) [15]. Fifth, differences in bioavailability result from differences in the concentrations and composition of particulate and dissolved organic matter in the water between laboratory conditions and field conditions. Finally, differences in size, lipid content, feeding habits, and growth rates among organisms can cause significant differences between laboratory and field derived water-internal concentration relationships.

Our results illustrate the difficulty with using exposure-based concentrations as a surrogate for toxicity, a difficulty that has also been pointed out by McCarty and Mackay [16] and others [17–20]. Using guidelines that are based on internal rather than external (e.g., water or sediment) concentrations is an advantage because they can ignore the issue of how chemicals are taken up from the water. However, the current water and sediment quality guideline development process has its merits, for example, in developing permits for waste water discharge, ocean dumping, and site remediation. In terms of improving the WQG development process to ensure consistency between water quality guidelines and internal tissue-based guidelines on a site-specific basis, the following suggestions can be made. First, as our results support, bioaccumulation models can be used when developing and applying environmental quality guidelines. In the absence of empirical data under all relevant conditions, these models can play an important role in capturing and quantifying differences that might exist between laboratory and field conditions as well as between various field conditions due to temporal and spatial factors and differences between chemical loading histories. Because of the costs associated with establishing statistically valid empirical data sets for guideline development, the use of established and well-tested food-chain models are ideal for the development of site-specific objectives. Second, it is important to develop more realistic safety factors when developing water quality guidelines. These safety factors should include potential uncertainties in both toxicokinetics and toxicodynamics. Third, the development of water quality and other environmental quality guidelines can be improved when standard toxicity experimental protocols, such as those developed by the Organization for Economic Cooperation Development and the American Society for Testing and Materials [21,22], include the measurement of the internal concentration as well as the exposure concentration associated with the toxic effect. Finally, internal-based guidelines would provide a basis for assessing mixtures of chemicals of similar modes of action [16].

Acknowledgement—We thank Randall Peterman of Simon Fraser University for statistical advice as well as helpful comments and sug-

gestions. The Natural Sciences and Engineering Research Council of Canada is acknowledged for financial support.

REFERENCES

1. Canadian Council of Ministers of the Environment. 1991. A protocol for the derivation of water quality guidelines for the protection of aquatic life. In *Canadian Water Quality Guidelines*, Appendix IX. Environmental Quality Branch, Environment Canada, Ottawa, ON.
2. Canadian Council of Ministers of the Environment. 1987. *Canadian Water Quality Guidelines*. Environmental Quality Branch, Environment Canada, Ottawa, ON, pp 1–8.
3. Spacie A, Hamelink JL. 1982. Alternative models for describing the bioconcentration of organics in fish. *Environ Toxicol Chem* 1:309–320.
4. Van Hoogen G, Opperhuizen A. 1988. Toxicokinetics of chlorobenzenes in fish. *Environ Toxicol Chem* 7:213–219.
5. Gobas FAPC. 1993. A model for predicting the bioaccumulation of hydrophobic organic chemicals in aquatic food webs: Application to Lake Ontario. *Ecol Model* 69:1–17.
6. DeFoe DL, Veith GD, Carlson RW. 1978. Effects of Aroclor (R) 1248 and 1260 on the fathead minnow (*Pimephales promelas*). *J Fish Res Board Can* 35:997–1002.
7. Smith AD, et al. 1991. The acute and chronic toxicity of ten chlorinated organic compounds to American flagfish (*Jordanella floridae*). *Arch Environ Contam Toxicol* 20:94–102.
8. Chiou CT. 1985. Partition coefficients of organic compounds in lipid-water systems and correlations with fish bioconcentration factors. *Environ Sci Technol* 19:57–62.
9. Smith AD, Bharath A, Mallard C, Orr D, McCarty LS, Ozburn GW. 1990. Bioconcentration kinetics of some chlorinated benzenes and chlorinated phenols in American flagfish, *Jordanella floridae* (Goode and Bean). *Chemosphere* 20:379–386.
10. Laseter JL, Bartell CK, Laska AL, Holmquist DG, Condie DB, Brown JW, Evans RL. 1976. An ecological study of hexachlorobenzene (HCB). EPA 560/6-76-009. PB-252 651. U.S. Environmental Protection Agency, Washington, DC.
11. Oliver BG, Niimi AJ. 1988. Trophodynamic analysis of polychlorinated biphenyl congeners and other chlorinated hydrocarbons in the Lake Ontario ecosystem. *Environ Sci Technol* 22: 388–397.
12. Niimi AJ, Oliver BG. 1989. Distribution of polychlorinated biphenyl congeners and other halocarbons in whole fish and muscle among Lake Ontario salmonids. *Environ Sci Technol* 23:83–88.
13. Spitsbergen JM, Walker MK, Olson JR, Peterson RE. 1991. Pathologic alterations in early life stages of lake trout, *Salvelinu namaycush*, exposed to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin as fertilized eggs. *Aquat Toxicol* 19:41–72.
14. Andersen DE, Singleton H, Spry D, Walker SL, Keenleyside KA, Kent RA, MacDonald D. 1995. Canadian environmental quality guidelines for dioxins and furans—A Multimedia approach. *Proceeding*, 21st Annual Aquatic Toxicity Workshop, Sarnia, ON, Canada, October 3–5, 1994, p 44.
15. Gobas FAPC, Z'Graggen MN. 1995. Time response of the Lake Ontario ecosystem to virtual elimination of PCBs. *Environ Sci Technol* 29:2038–2046.
16. McCarty LS, Mackay D. 1993. Enhancing ecotoxicological modelling and assessment: Body residues and modes of toxic action. *Environ Sci Technol* 27:1719–1727.
17. Landrum PF, Hayton WL, Lee H II, McCarty LS, Mackay D, McKim JM. 1994. Synopsis of discussion on the kinetics behind environmental bioavailability. In Hamelink JL, Landrum PF, Bergman HL, Benson WH, eds, *Bioavailability: Physical, Chemical and Biological Interactions*. Lewis, Boca Raton, FL, USA, pp 203–220.
18. McCarty LS, Mackay D, Smith AD, Ozburn GW, Dixon GW. 1992. Residue-based interpretation of toxicity and bioconcentration QSARs from aquatic bioassays: Neutral organic compounds. *Environ Toxicol Chem* 11:917–930.
19. Landrum PF, Lee H, Lydy MJ. 1992. Toxicokinetics in aquatic systems: Model comparisons and use in hazard assessment. *Environ Toxicol Chem* 11:1709–1725.
20. Gobas FAPC. 1992. Modelling the accumulation and toxicity of organic chemicals in aquatic food chains. In Gobas FAPC, McCorquodale JA, eds, *Chemical Dynamics in Fresh Water Ecosystems*. Lewis, Boca Raton, FL, USA, pp 129–153.
21. American Society for Testing and Materials. 1997. Standard guide for conducting early life-stage tests with fishes. E1241-92. In *Annual Book of ASTM Standards*, Vol 11.05—*Biological Effects and Environmental Fate; BioTechnology; Pesticides*. Philadelphia, PA, pp 550–576.
22. Organization for Economic Cooperation and Development. 1992. Fish, early-life stage toxicity tests. OECD Guideline for Testing of Chemicals 210. Paris, France.